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Original Research Communication

Low dietary magnesium increases supraventricular ectopy[1,2,3,4]

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ABSTRACT

Background: Magnesium has been suggested to be beneficial in counteracting all phases of the processes that lead to ischemic heart disease, including terminal events such as arrhythmia and sudden death.

Objective: We tested the hypothesis that an intake of magnesium considerably below the recommended dietary allowance can produce chemical and physiologic evidence of depletion.

Design: Twenty-two postmenopausal women were maintained in a metabolic unit and ate a diet of conventional foods containing less than one-half of or more than the recommended dietary allowance for magnesium (320 mg/d). Dietary assignments were random and double blind in a crossover design. Magnesium concentrations were measured by spectroscopy and ion-specific electrolyte analysis, and Holter electrocardiograms lasting ≈21 h were recorded.

Results: Magnesium concentrations in erythrocytes, serum (total and ultrafilterable), and urine were significantly lower when dietary magnesium was lower. Holter monitors showed a significant increase in both supraventricular and supraventricular plus ventricular beats when the dietary magnesium concentration was low. Hypomagnesemia, hypocalcemia, and hypokalemia were not found.

Conclusions: The magnesium requirement was defined with the use of biochemical and electrophysiologic criteria. The recommended dietary allowance of 320 mg/d seems correct; 130 mg is too little. Persons who live in soft water areas, who use diuretics, or who are predisposed to magnesium loss or ectopic beats may require more dietary magnesium than would others.

Key Words: Dietary requirements • ectopy • Holter monitor • soft water • magnesium • postmenopausal women • ischemic heart disease • arrhythmia • ectopic beats

INTRODUCTION

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Magnesium has been suggested to be beneficial in counteracting all phases of the harmful processes that lead to death from ischemic heart disease. For example, Selye (1, 2) mentioned magnesium as being protective against experimental cardiopathies. Seelig and Heggtveit (3) summarized some plausible mechanisms by which magnesium protects against cardiopathies. Hellerstein et al (4) found that a low-magnesium diet sometimes contributed to the production of aortic lipidosis in rats fed an atherogenic diet. Elin and Hosseini (5) suggested that magnesium may protect Seventh-day Adventists who eat nuts regularly from coronary heart disease. Chipperfield and Chipperfield (6) found less magnesium in the uninfarcted heart muscle of persons who died suddenly than in the heart muscle of healthy control subjects. Their data were related to the "water factor": ie, the risk of ischemic heart disease is higher where the drinking water is soft and lower where the drinking water is hard (7, 8). There is a high correlation between the magnesium content of water and the hardness of the water ($r = 0.90$) (9, 10). Other studies showed that intravenous magnesium seems beneficial in reducing the risk of arrhythmias immediately after myocardial infarction (11–15). More recent research on the possible benefits of magnesium in ischemic heart disease was directed toward cardiac electrophysiology (16–19) and the balance between supply and demand of the myocardium for oxygen (20).

According to Shils (21), magnesium deficiency in animals and people is accompanied by a variety of undesirable changes in neuromuscular function such as muscle fasciculations and spasm, tremor, tetany, and convulsions. Chvostek and Trousseau signs sometimes are present. Generally, these changes are

associated with hypomagnesemia, which also can be associated with hypocalcemia and hypokalemia. Serum magnesium concentration may not be a good indicator of systemic magnesium depletion (21–23); indeed, there are no reliable noninvasive techniques for determining magnesium status (24). Hypomagnesemia can increase the risk of supraventricular and ventricular arrhythmias, including ventricular tachycardia and ventricular fibrillation (25).

The recommended dietary allowance (RDA) for magnesium for women has been increased from 280 to 320 mg/d (and to >400 mg/d for men) (24, 26). These values are based on balance data and usual intakes of the US population. Magnesium intakes of women often are below the RDA (27). Committees that set RDAs generally err on the side of generosity (24), introducing a safety factor over requirements so that recommendations are adequate for practically all healthy persons.

We are engaged in long-standing research on measurements of mineral requirements to develop dietary recommendations to prevent disease, promote health, and allow optimal function throughout life. Thus, it is necessary to compare responses to lower and higher nutrient intakes. We tested the hypothesis that an intake of magnesium considerably below the RDA can produce both chemical and physiologic evidence of depletion. It seemed prudent and desirable to include frequent measurements of neuromuscular and cardiovascular functions in an experiment with 2 intakes of magnesium so that both safety and science could be served. This work is the only measurement of the magnesium requirement of healthy persons; it was planned and most data were collected before the recent RDA revision.

SUBJECTS AND METHODS

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Twenty-nine healthy, postmenopausal women aged 47–78 y ($\bar{x}$ ± SD: 64.7 ± 8.4 y) were admitted to the study after medical, psychological, and nutritional evaluations established that they had no underlying disease and were emotionally suited for the project. They were informed in detail of the nature of the research and associated risks. Protocols were approved by the Institutional Review Boards of the University of North Dakota and the US Department of Agriculture and followed the guidelines of the US Department of Health and Human Services and the Helsinki Declaration of 1975 as revised in 1983.

The women were maintained in a metabolic unit in groups of 15 and 14 and were under close supervision for ≈6 mo. Because dietary magnesium and copper are highly correlated (28), a diet designed to be low in magnesium also will be low in copper. Preliminary estimates made before the study began showed that the study diet (**Table 1**) was low in copper and magnesium, <1 and 100 mg/d, respectively, per 8.4 MJ. During a 10-d equilibration period, the diet was supplemented with 1.0 mg Cu and 200 mg Mg daily. During this period the women were assigned to 2 dietary groups in a random, double-blind, crossover design. According to group assignment, the women were then given the study diet either without a magnesium supplement (placebo) or with a magnesium supplement for 81 d. Then, women receiving the unsupplemented diet received the supplemented diet and vice versa for 81 more days. Diets of one-half of the women were supplemented with 1 mg/d Cu and the rest with 3 mg/d Cu

throughout the 162 d, with no crossover. Magnesium was supplemented as magnesium gluconate capsules (Willner Chemists, Inc, New York) in divided doses with meals, and copper was added as copper sulfate in juice. Placebo capsules contained lactose (Gallipot, Inc, St Paul).

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TABLE 1 Composition of the diet[1]

Diets were weighed carefully and were eaten completely (without any extra foods) in a 3-d rotation. The dietary intake of each subject was based on energy needs, as calculated by the Harris and Benedict equation (29), plus an additional 50% of basal energy expenditure for normal activity. During the study, energy intakes were adjusted to maintain body weight to within ±2% of admission weight by adjusting the amount of the basal diet in 837-kJ (200-kcal) increments.

Copper and magnesium in 6-d composites of diets were measured by inductively coupled argon plasma emission spectroscopy (ICAP; Perkin-Elmer, Norwalk, CT) after wet digestion of aliquots of freeze-dried material with nitric and perchloric acids (30). Urinary copper, magnesium, and calcium were determined by ICAP analysis of a diluted urine aliquot; values for the last 3 balance periods for each diet were averaged.

Blood was drawn into plastic syringes from an antecubital vein, which had been made visible by temporary use of a tourniquet, after the subjects had fasted for 10 h. Aliquots were processed within 90 min of the time the blood was drawn. Serum magnesium and calcium concentrations were determined by flame atomic absorption spectrophotometry after dilution 50-fold with an acidic lanthanum chloride diluent (31). Potassium, ionized calcium, and ionized magnesium were determined in heparinized plasma by using a NOVA CRT 8+ electrolyte analyzer (Nova Biomedical, Waltham, NJ). Serum ultrafilterable magnesium samples were prepared by using an Amicon MPS-1 filter system (Amicon, Inc, Beverly, MA) and a procedure described by D'Costa and Cheng (32). The magnesium content of the ultrafiltrate was determined by ICAP analysis (30). Parathyroid hormone in serum was determined by radioimmunoassay (Incstar, Stillwater, MN). Blood values are the mean of 2 values measured within 2 wk of the end of each dietary period.

Holter electrocardiograms were recorded with increasing frequency near the end of each dietary period (Del Mar Avionics, Irvine, CA). Data from the last 3 recordings evaluated by computerized scoring at the end of each dietary period were analyzed by repeated-measures analysis of variance after square root transformation (33). Chvostek and Trousseau signs were sought by, respectively, gently tapping the subject's cheek superficially over the facial nerve for 1–2 s and examining the subject's hand after her arm was encased in a sphygmomanometer cuff inflated to 10 mm Hg higher than systolic blood pressure for 2 min.

Although the intakes of copper were greater than the highest intake proved insufficient for women (34),

all cardiograms were examined for ventricular premature discharges and heart block because these changes have been detected in copper depletion studies (35, 36). If ventricular discharges were found to be 4-fold higher than the higher number found on 2 recordings during the equilibration period, copper supplements were given to women consuming 1 mg Cu/d because Velebit et al (37) found this increase to be evidence of aggravation of arrhythmias. Institutional review boards and volunteers had been promised copper supplements under these circumstances.

Statistical analysis was by repeated-measures analysis of variance (33).

RESULTS

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Replicate analysis of a total diet standard reference material (Total Diet SRM#1548; National Institute of Standards and Technology, Gaithersburg, MD) (n = 6) yielded values of 2.6 ± 0.14 and 550 ± 16 μg/g ($\bar{x}$ ± SD) compared with certified values of 2.3–2.9 and 540–572 μg/g for copper and magnesium, respectively, and concurrent analysis of a diet pool (n = 11) yielded values of 0.76 ± 0.06 μg/g and 0.26 ± 0.02 mg/g compared with the expected ranges of 0.63–0.83 μg/g and 0.23–0.30 mg/g for copper and magnesium, respectively. Direct chemical analysis determined that the women's mean, daily dietary magnesium intakes were 130 mg with the deficient diet and 411 mg with the supplemented diet.

Twenty-two women completed the 172-d study and contributed to the means mentioned in the text and tables. Three women resigned and 4 in the group receiving the lower amount of dietary copper were supplemented with extra dietary copper because of new cardiographic findings resembling those found in studies of copper depletion. One woman had a right bundle branch block, 1 woman had a few episodes of ventricular ectopy and atrioventricular dissociation, and 2 women had a >4-fold increase in ventricular discharges (to a maximum of 30 and 238 per 21 h). No one else in the study exhibited either heart block or a 4-fold increase in ventricular discharges. Neither feeding sequence nor copper effects were significant ($P > 0.1$).

Because the results were unaffected by dietary copper and by the sequence of the different intakes of magnesium, according to analysis of variance, data are presented only as to the intake of magnesium. Serum magnesium concentrations were significantly lower when the amount of magnesium in the diet was lower (**Table 2**) (3.5%), as were concentrations of erythrocyte magnesium (3.3%) and ultrafilterable magnesium in serum (4.2%). Inferences about erythrocyte magnesium were unaffected when the results were expressed per unit of cells or packed cells instead of per unit of hemoglobin. Urinary magnesium concentrations were 49% lower when the amount of magnesium in the diet was lower. No significant changes were noted in potassium, ionized calcium, or ionized magnesium concentrations in plasma or in serum calcium or parathyroid hormone. Neither Chvostek nor Trousseau sign was positive in any of 25 weekly examinations. Daily loss of magnesium via feces and urine exceeded dietary intake by 3.9 mmol (94 mg) (P = 0.0001) when the amount of dietary magnesium was low (data not shown).

View this table: [in this window] [in a new window] **TABLE 2** Magnesium measurements[1]

Holter electrocardiograms were recorded for a mean of 20.7 ± 0.87 h and 84990 ± 10595 heartbeats. Data on ectopy are summarized in **Table 3**. Supraventricular beats were more frequent when dietary magnesium was low. Although the increase in ventricular ectopy was not significant, the sum of supraventricular plus ventricular beats was significantly higher when dietary magnesium was low. One woman had one episode of 4 consecutive ventricular beats. No other ventricular tachycardia or ventricular fibrillation was detected during 130 recordings.

View this table: [in this window] [in a new window] **TABLE 3** Ectopy[1]

DISCUSSION

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The diet used in this study was both ordinary and apparently adequate. Its inadequacy in magnesium was shown by biochemical and physiologic changes. Restriction of magnesium intake resulted in decreased urinary loss, as reported by Shils (21), who mentions low urinary magnesium as an early sign of depletion. The mean loss in depletion was slightly lower than the minimal daily loss assumed to be normal (38, 39). Magnesium concentrations decreased in serum and erythrocytes, but the hypomagnesemia, hypocalcemia, and hypokalmia Shils reported were not found in our study, probably because the intake of magnesium here was considerably higher than in his studies. Parathyroid hormone concentrations did not increase, probably because serum calcium and ionized calcium concentrations did not decrease; perhaps the decrease in serum magnesium concentration counteracted changes in calcium too subtle to be detected.

Women in this study consumed a diet low in magnesium for 81 d. Decreases in magnesium indexes showed that the women were being depleted of magnesium. A longer experiment or a diet lower in magnesium might have produced greater changes, with values in Table 2 moving into the deficiency range.

Although none of the mean blood values in Table 2 were below respective normal ranges, 130 mg Mg/d was sufficiently low to modify magnesium homeostasis. Because 99% of magnesium in the body is intracellular (21, 24), assessment of magnesium status is difficult (21–23). The decrease in erythrocyte magnesium concentrations may reflect intracellular magnesium in myocardial cells.

A magnesium-dependent ATPase controls sodium and potassium transport across cell membranes (40). Impairment of this pump as a result of a low intake of dietary magnesium may have altered membrane potential and myocardial irritability. Magnesium is central to a variety of cellular mechanisms that control activity of muscle and nerve cells (41). Cardiac muscle seems to have been more sensitive to this intake than was skeletal muscle, as shown by the absence of Chvostek and Trousseau signs in the present study.

The frequency of supraventricular beats was higher with lower amounts of dietary magnesium. Perhaps if a larger group of women were exposed to these diets, the rate of ventricular ectopy also would increase significantly. Although the standard settings of the instrument reflected widely used clinical criteria, the criteria discriminating between the 2 types of ectopy are somewhat arbitrary. Because of this uncertainty, we calculated the combined frequency of ectopy. Results were unaffected by dietary copper. Magnesium depletion was verified by both chemical and physiologic criteria.

An intake of magnesium of less than one-half of the RDA changed cardiac rhythm. The daily dietary magnesium requirement of these middled-aged women exceeded 130 mg/d; 411 mg daily was sufficient. A criterion based on cardiac electrophysiology confirmed the correctness of the RDA. Whether extra dietary magnesium will decrease idiopathic ectopy is worth testing.

Some women may habitually eat a diet similar to that in this experiment for long periods. According to Pennington (27), magnesium intakes of women in the United States generally do not meet the 1989 RDA of 280 mg; some groups average <75% of this amount, or 210 mg/d. If magnesium intakes are positively skewed similarly to those of copper (42), substantial numbers of women must eat 130 mg daily with some regularity. This amount is ≈40% of the revised RDA of 320 mg (26).

The increase in the number of ecoptic beats in this experiment was both small, ≈100/d, and undesirable. Extra systoles are common in healthy persons (43) and generally are of unknown origin. Whether a diet low in magnesium contributes to this phenomenon is unknown; however, a diet low in magnesium probably will not benefit persons with valvular disease or cardiac hypertrophy or persons who consume toxins such as alcohol or caffeine, which predispose to this arrhythmia. These persons may require more dietary magnesium than average. Leddingham and Raine (25) suggest that magnesium depletion may occur with the use of diuretics, especially in subjects whose dietary intake is low and who live in soft water areas. Shils (21) listed several clinical conditions contributing to magnesium depletion via decreased absorption or increased loss or with endocrine disorder. Perhaps patients with enteropathies from atrophy, gluten, inflammation, infection, radiation, or surgery; renal dysfunction from nephrotoxic drugs or tubular diseases; endocrine disorders such as diabetes mellitus, hyperaldosteronism, or hyperthyroidism; or familial disorders such as Bartter syndrome or renal wasting syndrome will be more at risk from a diet low in magnesium than others.

In summary, an apparently adequate, ordinary diet low in magnesium disrupted magnesium homeostasis. An increase in myocardial irritability was detected with Holter electrocardiography.

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REFERENCES

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1. Selye H. The pluricausal cardiopathies. Springfield, IL: Charles C Thomas, 1961.
2. Selye H. Experimental cardiovascular diseases. New York: Springer-Verlag, 1970.
3. Seelig MS, Heggtveit HA. Magnesium interrelationships in ischemic heart disease: a review. Am J Clin Nutr 1974;27:59–79.[Medline]
4. Hellerstein EE, Nakamura M, Hegsted DM, Vitale JJ. Studies on the interrelationships between dietary magnesium, quality and quantity of fat, hypercholesterolemia and lipidosis. J Nutr 1960;71:339–46.
5. Elin RJ, Hosseini JM. Is the magnesium content of nuts a factor for coronary heart disease? Arch Intern Med 1993;153:779–80.
6. Chipperfield B, Chipperfield JR. Differences in metal content of the heart muscle in death from ischemic heart disease. Am Heart J 1978;95:732–7.[Medline]
7. Klevay LM. The influence of copper and zinc on the occurrence of ischemic heart disease. J Environ Pathol Toxicol 1980;4:281–7.[Medline]
8. Lacey RF, Shaper AG. Changes in water hardness and cardiovascular death rates. Int J Epidemiol 1984;13:18–24.[Abstract]
9. Klevay LM. Coronary heart disease: the zinc/copper hypothesis. Am J Clin Nutr 1975;28:764–74. [Abstract]
10. Klevay LM. Ischemic heart disease: toward a unified theory. In: Lei KY, Carr TP, eds. Role of copper in lipid metabolism. Boca Raton, FL: CRC Press, 1990:233–67.
11. Rasmussen HS, McNair P, Norregard P, Backer V, Lindeneg O, Balslov S. Intravenous magnesium in acute myocardial infarction. Lancet 1986;1:234–6.[Medline]
12. Abraham AS, Rosenmann D, Kramer M, et al. Magnesium in the prevention of lethal arrhythmias in acute myocardial infarction. Arch Intern Med 1987;147:753–5.[Abstract]
13. Ceremuzynski L, Jurgiel R, Kulakowski P, Gebalska J. Threatening arrhythmias in acute myocardial infarction are prevented by intravenous magnesium sulfate. Am Heart J 1989;118:1333–4.[Medline]
14. Teo KK, Yusuf S, Collins R, Held PH, Peto R. Effects of intravenous magnesium in suspected acute myocardial infarction: overview of randomised trials. BMJ 1991;303:1499–503.[Medline]
15. Gurfinkel E, Pazos A, Mautner B. Abnormal QT intervals associated with negative T waves induced by antiarrhythmic drugs are rapidly reduced using magnesium sulfate as an antidote. Clin Cardiol 1993; 16:35–8.[Medline]
16. Lasserre B, Spoerri M, Moullet V, Theubet MP. Should magnesium therapy be considered for the treatment of coronary heart disease? II. Epidemiological evidence in outpatients with and without coronary heart disease. Magnes Res 1994;7:145–53.[Medline]
17. Weiss M, Lasserre B. Should magnesium therapy be considered for the treatment of coronary heart disease? I. A critical appraisal of current facts and hypotheses. Magnes Res 1994;7:135–44. [Medline]
18. Zaman AG, Alamgir F, Richens T, Williams R, Rothman MT, Mills PG. The role of signal

averaged P wave duration and serum magnesium as a combined predictor of atrial fibrillation after elective coronary artery bypass surgery. Heart 1997;77:527–31.[Abstract]
19. Baraka A, Ayoub C, Kawkabani N. Magnesium therapy for refractory ventricular fibrillation. J Cardiothorac Vasc Anesth 2000;14:196–9.[Medline]
20. Vigorito C, Giordano A, Ferraro P, et al. Reduction of pacing-induced myocardial ischemia by intravenous magnesium sulfate. Am J Cardiol 1995;75:280–2.[Medline]
21. Shils ME. Magnesium. In: Ziegler EE, Filer LJ Jr, eds. Present knowledge in nutrition. 7th ed. Washington, DC: ILSI Press, 1996:256–64.
22. Milne DB. Trace elements. In: Burtis CA, Ashwood ER, eds. Tietz textbook of clinical chemistry. 3rd ed. Philadelphia: WB Saunders, 1999:1029–55.
23. Milne DB. Laboratory assessment of trace element and mineral status. In: Bogden JD, Klevay LM, eds. Clinical nutrition of the essential trace elements and minerals: the guide for health professionals. Totowa, NJ: Humana Press, 2000:69–90.
24. National Research Council. Recommended dietary allowances. 10th ed. Washington, DC: National Academy Press, 1989.
25. Ledingham JG, Raine AE. The treatment of heart failure: diuretics. In: Weatherall DJ, Ledingham JG, Warrell DA, eds. Oxford textbook of medicine. 3rd ed. New York: Oxford University Press, 1996: 2238–41.
26. Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, Food and Nutrition Board, Institute of Medicine. Dietary reference intakes for calcium, phosphorus, magnesium, vitamin D, and fluoride. Washington, DC: National Academy Press, 1997.
27. Pennington JA. Current dietary intakes of trace elements and minerals. In: Bogden JD, Klevay LM, eds. Clinical nutrition of the essential trace elements and minerals: the guide for health professionals. Totowa, NJ: Humana Press, 2000:49–67.
28. Klevay LM. Magnesium, calcium, copper, and zinc in meals. Correlations related to the epidemiology of ischemic heart disease. Biol Trace Elem Res 1982;4:95–104.
29. Harris JA, Benedict FG. A biometric study of basal metabolism in man. Philadelphia: JB Lippencott, 1919.
30. Sims RL, Mullen LM, Milne DB. Application of inductively coupled plasma emission spectroscopy to multielement analysis of foodstuffs used in metabolic studies. J Food Comp Anal 1990;3:27–37.
31. Bowers GN Jr, Rains TC. Measurement of total calcium in biological fluids: flame atomic absorption spectrometry. Methods Enzymol 1988;158:302–19.[Medline]
32. D'Costa M, Cheng PT. Ultrafiltrable calcium and magnesium in ultrafiltrates of serum prepared with the Amicon MPS-1 system. Clin Chem 1983;29:519–22.[Abstract/Free Full Text]
33. Winer BJ. Statistical principles in experimental design. 2nd ed. New York: McGraw-Hill, 1971.
34. Lukaski HC, Klevay LM, Milne DB. Effects of dietary copper on human autonomic cardiovascular function. Eur J Appl Physiol 1988; 58:74–80.
35. Klevay LM, Inman L, Johnson LK, et al. Increased cholesterol in plasma in a young man during experimental copper depletion. Metabolism 1984;33:1112–8.[Medline]
36. Reiser S, Smith JC Jr, Mertz W, et al. Indices of copper status in humans consuming a typical American diet containing either fructose or starch. Am J Clin Nutr 1985;42:242–51.[Abstract]
37. Velebit V, Podrid P, Lown B, Cohen BH, Graboys TB. Aggravation and provocation of ventricular arrhythmias by antiarrhythmic drugs. Circulation 1982;65:886–94.[Abstract]
38. Tietz NW, ed. Clinical guide to laboratory tests. 3rd ed. Philadelphia: WB Saunders, 1995.
39. Giles AM, Holoway P. Reference intervals for biochemical data. In: Weatherall DJ, Ledingham JG, Warrell DA, eds. Oxford textbook of medicine. 3rd ed. New York: Oxford University Press, 1996:4363–76.
40. Opie LH. The heart: physiology and metabolism. 2nd ed. New York: Raven Press, 1991.
41. Strata P. Magnesium and the cell membrane. In: Vecchiet L, ed. Magnesium & physical activity. New York: Parthenon Publishing Group, 1995:83–92.

42. Klevay LM. Lack of a recommended dietary allowance for copper may be hazardous to your health. J Am Coll Nutr 1998;17:322–6.[Abstract/Free Full Text]
43. Cobbe SM, Rankin AC. Cardiac arrhythmias. In: Weatherall DJ, Ledingham JG, Warrell DA, eds. Oxford textbook of medicine. 3rd ed. New York: Oxford University Press, 1996:2259–85.

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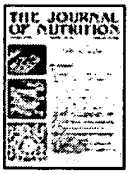

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